Attachment 2. Cadmium in Blood—Half life or pharmacokinetics From Medline

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Medline Search Strategy
Set     Items   Description
S2      24950   "HALF-LIFE"
S3       7153   CADMIUM/TI
S4         82   S3 AND (S1 OR S2)
S5        135   "CADMIUM POISONING --PATHOLOGY --PA"
S6          0   S5.MAJ
S7         79   S5/MAJ
S8         73   S3 AND S7
S9          9   S8/ENG, HUMAN
S10        47   S2 AND S3
S11       624   "CADMIUM --BLOOD --BL"
S12       541   "CADMIUM --PHARMACOKINETICS --PK"
S13        11   S2 AND S12
S14        24   S11 AND S12
S15       144   S8 OR S10 OR S13 OR S14
S16        33   S15/ENG,HUMAN
? t s16/9/1-33

 16/9/1
DIALOG(R)File 155:MEDLINE(R)
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10373335   20187909
  Metal-bone interactions.
  Berglund M; Akesson A; Bjellerup P; Vahter M
  Institute of Environmental Medicine, Box 210, S-171 77, Stockholm,
Sweden. marika.beglud@imm.ki.se
  Toxicology letters (NETHERLANDS) Mar 15 2000, 112-113 p219-25, ISSN
0378-4274   Journal Code: VXN
  Languages: ENGLISH
  Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
  JOURNAL ANNOUNCEMENT: 0007
  Subfile:   INDEX MEDICUS
  Recent studies indicate that lead and cadmium may exert both direct and
indirect actions on bone turnover, indirectly via kidney dysfunction, and
directly on osteoblast and osteoclast function. Increased blood lead
concentrations, most likely as a result of an increased bone turnover, have
been detected in pregnant, lactating, and menopausal women. Lead exposure
has also been negatively associated with children's growth in stature. Both
lead and cadmium are nephrotoxic and can disturb vitamin D metabolism.
Cadmium has been shown to induce kidney damage and
osteoporosis/osteomalacia at long-term high-level exposure. A negative
association between cadmium dose and bone mass has recently been detected
in both occupationally and environmentally exposed people at relatively low
cadmium exposure. (41 Refs.)
  Tags: Female; Human; Male
  Descriptors: *Bone Remodeling--Drug Effects--DE; *Cadmium; *Lead; Adult;
Bone Density--Drug Effects--DE; Cadmium--Adverse Effects--AE; Cadmium
--Pharmacokinetics--PK; Calcium--Metabolism--ME; Half-Life; Homeostasis
--Drug Effects--DE; Kidney--Drug Effects--DE; Kidney--Metabolism--ME; Lead
--Adverse Effects--AE; Lead--Blood--BL; Lead--Pharmacokinetics--PK;
Middle Age; Pregnancy; Vitamin D--Metabolism--ME
  CAS Registry No.: 1406-16-2 (Vitamin D); 7439-92-1 (Lead); 7440-43-9
 (Cadmium); 7440-70-2 (Calcium)
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16/9/2
DIALOG(R)File 155:MEDLINE(R)

10348949 20169402
Cadmium-induced acute hepatic injury is exacerbated in human interleukin-8 transgenic mice.
Horiguchi H; Harada A; Oguma E; Sato M; Homma Y; Kayama F; Fukushima M; Matsushima K
Department of Public Health, Faculty of Medicine, Fukushima Medical University, 1 Hikarigaoka, Fukishima, 960-1295, Japan. hhyogo@jichi.ac.jp
Toxicology and applied pharmacology (UNITED STATES) Mar 15 2000, 163 (3) p231-9, ISSN 0041-008X Journal Code: VWO
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 0006
Subfile: INDEX MEDICUS
It is reported repeatedly that severe hepatocellular necrosis along with infiltration of neutrophils occurs after acute cadmium exposure. Neutrophils, which migrate by the gradient of chemoattractants such as interleukin-8, are believed to play an important role in inflammation at the damaged sites. To investigate whether neutrophils aggravate or repair the liver injury induced by cadmium, we checked the hepatotoxic effects of cadmium on human interleukin-8 transgenic mice (hIL-8Tg), which overexpressed IL-8 and displayed an inability of neutrophil migration resulting from both the lack of chemotactic gradient and the downregulation of l-selectin on the surface of neutrophils. A significantly lower survival rate was observed in hIL-8Tg compared with wild-type mice after subcutaneous administration of cadmium. Evident liver injury characterized by abrupt increases in plasma GOT and GPT levels was found in hIL-8Tg at 18 h after cadmium administration. Histological examinations, including H & E staining and esterase staining, revealed the infiltration of numerous neutrophils into the damaged liver tissues in wild-type mice, and the inhibition of the neutrophil migration into the liver as well as enhanced hepatocellular necrosis in hIL-8Tg. Peripheral white blood cell and polymorphonuclear cell counts increased and reached their peaks at 12 h after cadmium administration in wild-type mice, whereas the increase in blood leukocyte counts was delayed in hIL-8Tg. There was no significant difference in the amounts of cadmium accumulated in liver and kidneys between wild-type mice and hIL-8Tg. In conclusion, an acute cadmium hepatotoxic effect was exacerbated in hIL-8Tg resulting from inhibited neutrophil migration, suggesting that migrated neutrophils can prevent aggravation of liver injury by acute cadmium administration. Copyright 2000 Academic Press.
Tags: Animal; Human; Male; Support, Non-U.S. Gov't
Descriptors: *Cadmium Poisoning--Pathology--PA; *Hepatitis, Toxic --Pathology--PA; *Interleukin-8--Physiology--PH; Alanine Transaminase --Blood--BL; Aspartate Transaminase--Blood--BL; Cadmium--Pharmacokinetics --PK; Cadmium Poisoning--Mortality--MO; Hepatitis, Toxic--Mortality--MO; Interleukin-8--Biosynthesis--BI; Kidney--Metabolism--ME; Kidney--Pathology --PA; Leukocyte Count--Drug Effects--DE; Liver--Enzymology--EN; Liver --Metabolism--ME; Liver--Pathology--PA; Metallothionein--Blood--BL; Mice; Mice, Inbred BALB C; Mice, Transgenic; Neutrophil Infiltration--Drug Effects--DE; Reverse Transcriptase Polymerase Chain Reaction; Selectins --Biosynthesis--BI; Survival Analysis
CAS Registry No.: 0 (Interleukin-8); 0 (Selectins); 7440-43-9 (Cadmium); 9038-94-2 (Metallothionein)
Enzyme No.: EC 2.6.1.1 (Aspartate Transaminase); EC 2.6.1.2 (Alanine Transaminase)

16/9/3
DIALOG(R)File 155:MEDLINE(R)

09890799 99190275

Cadmium: a possible etiological factor in peripheral polyneuropathy.

Viaene MK; Roels HA; Leenders J; De Groof M; Swerts LJ; Lison D; Masschelein R

Department of Occupational Medicine, Catholic University of Leuven, Belgium. leenders.viaene@village.uunet.be

Neurotoxicology (UNITED STATES) Feb 1999, 20 (1) p7-16, ISSN 0161-813X Journal Code: OAP

Languages: ENGLISH

Document type: CLINICAL TRIAL; JOURNAL ARTICLE; RANDOMIZED CONTROLLED TRIAL

JOURNAL ANNOUNCEMENT: 9907

Subfile: INDEX MEDICUS

Uncovering the exact cause of polyneuropathies seems to be impossible in up to 24% of the cases. Experimental studies have shown that cadmium (Cd), which is a well-known occupational and environmental hazard, can be a potent neurotoxicant for the peripheral nervous system. Moreover, Cd has a half-life of more than 15 years in humans. We hypothesize that older workers may be more susceptible to an increased Cd body burden, and may develop a peripheral polyneuropathy (PNP) over time. A blinded epidemiological survey was performed in 13 retired, long-term Cd-exposed workers and 19 age-matched controls. Historical Cd biomonitoring data were available over the last two decades. A neurological clinical examination, nerve conduction studies, and needle EMG were performed, and a standardized questionnaire was given to evaluate polyneuropathy complaints. If two of the following four criteria, i.e. complaints of polyneuropathy, neurophysiological changes compatible with polyneuropathy, distal symmetrical areflexia, or distal symmetrical anesthesia for vibration sense, temperature or blunt-sharp discrimination were present, the diagnosis of PNP was made. Two (11%) of the control and seven (54%) of the retired Cd workers met the PNP criteria OR: 9.92 (95%CI 1.60-61.6), Fisher exact test p=0.015. The existence of a polyneuropathy was related to the level of the Cd body burden as reflected by urinary Cd multiple logistic regression p=0.016, OR=1.26, (95%CI, 1.04-1.51), but not to blood lead (p=0.352). Our findings favour the hypothesis of a promoting role of increased cadmium body burden in the development of PNP at older age.

Tags: Human; Male; Support, Non-U.S. Gov't

Descriptors: *Cadmium Poisoning--Pathology--PA; *Occupational Exposure --Adverse Effects--AE; *Peripheral Nervous System Diseases--Etiology--ET; Aged; Autonomic Nervous System Diseases--Chemically Induced--CI; Autonomic Nervous System Diseases--Pathology--PA; Body Burden; Cadmium Poisoning --Epidemiology--EP; Cross-Sectional Studies; Dose-Response Relationship, Drug; Double-Blind Method; Electromyography; Follow-Up Studies; Middle Age; Neurologic Examination; Neuropsychological Tests; Peripheral Nervous System Diseases--Epidemiology--EP; Peripheral Nervous System Diseases--Pathology --PA; Questionnaires

16/9/4
DIALOG(R)File 155:MEDLINE(R)

08635980 96191005
Bioavailability of cadmium from shellfish and mixed diet in women.
Vahter M; Berglund M; Nermell B; Akesson A
Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden.
Toxicology and applied pharmacology (UNITED STATES) Feb 1996, 136 (2) p332-41, ISSN 0041-008X Journal Code: VWO
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9608
Subfile: INDEX MEDICUS
Dietary intake and uptake of cadmium (Cd) were studied in nonsmoking women, 20-50 years of age, consuming a mixed diet low in shellfish (N = 34) or with shellfish once a week or more (N = 17). Duplicate diets were collected during 4 consecutive days for the determination of Cd content. The women kept detailed dietary records, and the intake of energy and various nutrients was calculated. The shellfish diets (median 22.3 micrograms Cd/day) contained twice as much Cd as the mixed diets (median 10.5 micrograms Cd/day; $p < 0.0001$). Cadmium in feces corresponded to 100 and 99% of that in duplicates of shellfish diets and mixed diets, respectively, indicating a low average absorption of the dietary Cd. In spite of the differences in the daily intake of Cd, there was no statistically significant difference in the concentrations of Cd in blood (B-Cd, shellfish group 0.25 micrograms/liter, mixed diet group 0.23 micrograms/liter) or urine (U-Cd, 0.10 micrograms Cd/liter in both groups). This indicates a lower absorption of Cd in the shellfish group than in the mixed diet group or a difference in the kinetics. A higher gastrointestinal absorption of Cd in the mixed diet group could partly be explained by lower body iron stores as measured by the concentrations of serum ferritin (S-fer, median 18 micrograms/liter, compared to 31 micrograms/liter in the shellfish group). In the mixed diet group, S-fer was negatively correlated with B-Cd and the main determining for B-Cd besides U-Cd in the multiple regression analysis, indicating an increased absorption of Cd at low body iron stores. When women with S-fer exceeding 20 micrograms/liter were compared, the higher dietary intake of Cd in the shellfish group compared to the mixed diet group (24 versus 10 micrograms/day) resulted in higher B-Cd (0.26 versus 0.16 micrograms/liter), although not in proportion to the difference in Cd intake. Thus, there seems to be differences in the bioavailability and/or kinetics of dietary Cd related to the type of diet. This is, to our knowledge, the first study where the influence of various types of diets and nutritional factors on the intake and uptake of cadmium in human subjects has been studied.
Tags: Female; Human; Support, Non-U.S. Gov't
Descriptors: *Cadmium--Pharmacokinetics--PK; *Diet; *Shellfish; Adult; Biological Availability; Cadmium--Administration and Dosage--AD; Cadmium--Blood--BL; Cadmium--Urine--UR; Eating; Feces--Chemistry--CH; Ferritin--Blood--BL; Hemoglobins--Analysis--AN; Intestinal Absorption; Linear Models; Middle Age; Questionnaires; Zinc--Blood--BL
CAS Registry No.: 0 (Hemoglobins); 7440-43-9 (Cadmium); 7440-66-6 (Zinc); 9007-73-2 (Ferritin)

16/9/5
DIALOG(R)File 155:MEDLINE(R)

08296078 95273941
Lead and cadmium levels in human milk and blood.
Hallen IP; Jorhem L; Lagerkvist BJ; Oskarsson A
Toxicology Division Swedish National Food Administration, Uppsala.
Science of the total environment (NETHERLANDS) Apr 21 1995, 166 p149-55, ISSN 0048-9697 Journal Code: UJ0
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9508
Subfile: INDEX MEDICUS
Lead and cadmium levels were determined (with AAS) in blood and milk obtained at 6 weeks after delivery from women living in the vicinity of a copper and lead metal smelter and in a control area. Analysis of lead and cadmium were also performed in blood samples obtained at delivery. Accuracy of the analysis was confirmed by the analysis of quality control samples. In general, blood and milk levels of lead and cadmium were low in both areas. At 6 weeks after delivery the lead levels in blood and milk were 32 +/- 8 and 0.7 +/- 0.4 micrograms Pb/l, respectively (total mean +/- S.D., n = 75). Cadmium levels in blood and milk were 0.9 +/- 0.3 and 0.06 +/- 0.04 microgram Cd/l, respectively (n = 75). At delivery the lead levels in blood of women in the smelter area were higher, 38.7 micrograms Pb/l, than the blood lead levels in women from the control area, 32.3 micrograms Pb/l, (P < 0.001). At 6 weeks after delivery there was no difference in blood lead levels between the two groups. In contrast, the lead levels in milk were higher in women from the smelter area, 0.9 microgram Pb/l, than in women from the control area, 0.5 microgram Pb/l, (P < 0.001). No differences in blood cadmium levels were found between the two groups. Milk cadmium levels in women from the control area, 0.07 microgram Cd/l, were somewhat higher (P < 0.01) than in women from the smelter area, 0.05 microgram Cd/l.(ABSTRACT TRUNCATED AT 250 WORDS)
Tags: Female; Human; Support, Non-U.S. Gov't
Descriptors: *Cadmium--Pharmacokinetics--PK; *Environmental Pollutants --Pharmacokinetics--PK; *Lead--Pharmacokinetics--PK; *Milk, Human --Chemistry--CH; Analysis of Variance; Cadmium--Blood--BL; Chemical Industry; Environmental Pollutants--Blood--BL; Food Habits; Lactation; Lead --Blood--BL; Questionnaires; Smoking--Metabolism--ME; Spectrophotometry, Atomic Absorption; Sweden
CAS Registry No.: 0 (Environmental Pollutants); 7439-92-1 (Lead); 7440-43-9 (Cadmium)

16/9/6
DIALOG(R)File 155:MEDLINE(R)

08279768 95246698

Estimation of variation among individuals of biological half-time of cadmium calculated from accumulation data.

Sugita M; Tsuchiya K

Department of Environmental and Occupational Health, Toho University School of Medicine, Tokyo, Japan.

Environmental research (UNITED STATES) Jan 1995, 68 (1) p31-7, ISSN 0013-9351 Journal Code: EI2

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9508

Subfile: INDEX MEDICUS

For heavy metals, many experimental studies using animals have revealed short biological half-times (BHTs) to be 1 year or less by administration of heavy metals. Tsuchiya and Sugita, however, devised an original method and first reported the possibility of a long BHT for cadmium (Cd) calculated from Cd accumulation in postmortem human organs and tissues by age using a nonlinear regression analysis employing a differential equation. According to their reports, the estimated values of Cd BHT were 12.1-22.7 years in the kidney. The estimated Cd BHTs are point estimators. The point estimator of Cd BHT has been used indiscriminately to derive safety levels for Cd in the air of work environments or foodstuffs in the general population without taking into consideration individual variation of Cd BHT. In the present study, the estimated variation among individuals of Cd BHT ranges from a few years to at least 100 years in the kidney. It is concluded that the estimated average BHT according to mathematical calculation should not be used indiscriminately to derive the safety level of cadmium exposure.

Tags: Female; Human; Male

Descriptors: *Cadmium--Pharmacokinetics--PK; *Kidney--Metabolism--ME; Adolescence; Adult; Aged; Aged, 80 and over; Cadmium--Adverse Effects--AE; Cadmium--Analysis--AN; Child; Child, Preschool; Data Collection; Half-Life ; Infant; Infant, Newborn; Kidney--Chemistry--CH; Mathematics; Middle Age; Regression Analysis

CAS Registry No.: 7440-43-9 (Cadmium)

16/9/7
DIALOG(R)File 155:MEDLINE(R)

08257280 95198819
Morphometric studies of renal lesions in Itai-itai disease: chronic cadmium nephropathy.
Yasuda M; Miwa A; Kitagawa M
Department of Pathology, Toyama Medical and Pharmaceutical University, Faculty of Medicine, Japan.
Nephron (SWITZERLAND) 1995, 69 (1) p14-9, ISSN 0028-2766
Journal Code: NW8
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9506
Subfile: INDEX MEDICUS
Renal cortex in 15 cases with Itai-itai disease was morphometrically examined, i.e. the cross-size of tubuli in the renal cortex was measured using a color image analyzer (Olympus CIA-102) and the ratio of preserved urinary tubuli occupying renal cortex was calculated by means of the point-counting method. The former and the latter decreased gradually together as atrophy of kidney advanced. However, their decrease was stopped at about 60 g of kidney weight and it was inferred to be the limit of histopathologic changes of the kidneys with Itai-itai disease. Comparing the cross-size of the tubuli of the outer cortex area [= S(o)] with one of the inner cortex area [= S(i)], changes were more marked in S(o). This revealed that the outer cortex area was more sensitive to Cd intoxication and it was more affected by systemic atherosclerosis than the inner cortex area. It was concluded that when atrophy of kidneys as seen with Itai-itai disease was extremely progressing, decreases in the number and volume of tubuli in the cortex usually occurred at the same time and in a regular manner.
Tags: Comparative Study; Female; Human; Male
Descriptors: *Cadmium Poisoning--Pathology--PA; *Kidney--Drug Effects--DE ; *Kidney--Pathology--PA; Aged; Aged, 80 and over; Body Weight--Drug Effects--DE; Chronic Disease; Kidney--Anatomy and Histology--AH; Kidney Cortex--Drug Effects--DE; Kidney Cortex--Pathology--PA; Kidney Tubules --Drug Effects--DE; Kidney Tubules--Pathology--PA; Middle Age; Organ Weight--Drug Effects--DE

16/9/8
DIALOG(R)File 155:MEDLINE(R)

08027462 95020821
The cadmium toxicity hypothesis of aging: a possible explanation for the zinc deficiency hypothesis of aging.
Bin QH; Garfinkel D
Human Province, Ma-Wang-Dui Sanatorium, Chang Sha, PR China.
Medical hypotheses (ENGLAND) Jun 1994, 42 (6) p380-4, ISSN 0306-9877
Journal Code: M0M
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9501
Subfile: INDEX MEDICUS
Although cadmium and zinc have similar chemical properties, they affect living organisms diversely: while zinc is an essential element for growth, development and functioning of all living cells, cadmium is a highly toxic material. Cadmium has an extremely long biological half-life and may be considered a cumulative toxin. It has been shown to have sterilizing, teratogenic and carcinogenic effects and most of these effects could be reduced or even prevented by zinc administration. An increase in cadmium concentration with age has been proven in various species and in different tissues and these facts led some investigators to the assumption that cadmium accumulation might play an important role in senescence. Zinc essentiality and the lack of a reliable index of intracellular zinc status, formed the rationale for the zinc deficiency hypothesis of aging. This hypothesis suggests a gradual time related zinc deficiency occurring in each living cell, making zinc less available for its metalloenzymes. The sum of all deleterious effects resulting from the distorted function of different zinc enzymes, is later manifested as aging processes. When cadmium concentration increases, zinc concentration in various tissues decreases. Cadmium may inhibit zinc activities at many stages, interfering with zinc absorption, distribution to different tissues and transport into cells or into several intracellular structures. Therefore, it is reasonable to assume that a slowly developing cadmium toxicity may result in a gradual time related zinc deficiency.
Tags: Animal; Human
Descriptors: *Aging--Metabolism--ME; *Cadmium--Adverse Effects--AE; *Zinc --Physiology--PH; Cadmium--Pharmacokinetics--PK; Cadmium--Toxicity--TO; Cardiovascular Diseases--Chemically Induced--CI; Clinical Trials; Double-Blind Method; Half-Life; Lipid Peroxidation--Drug Effects--DE; Macular Degeneration--Prevention and Control--PC; Mutation--Drug Effects --DE; Neoplasms, Experimental--Chemically Induced--CI; Zinc--Deficiency--DF ; Zinc--Therapeutic Use--TU
CAS Registry No.: 7440-43-9 (Cadmium); 7440-66-6 (Zinc)

16/9/9
DIALOG(R)File 155:MEDLINE(R)

07946367 94282934

Cadmium: exposure markers as predictors of nephrotoxic effects.

Lauwerys RR; Bernard AM; Roels HA; Buchet JP

Industrial Toxicology and Occupational Medicine Unit, Catholic University of Louvain, Brussels, Belgium.

Clinical chemistry (UNITED STATES) Jul 1994, 40 (7 Pt 2) p1391-4, ISSN 0009-9147 Journal Code: DBZ

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

JOURNAL ANNOUNCEMENT: 9409

Subfile: INDEX MEDICUS

Cadmium (Cd) is a cumulative element with a biological half-life of > 10 years in humans. The total amount of Cd accumulated in the liver and in the kidney can be measured in vivo by neutron activation (or x-ray fluorescence), but this technique does not necessarily measure the fraction that is biologically active. At low exposure (i.e., general environmental exposure or moderate occupational exposure), blood Cd is mainly influenced by the last 2 to 3 months of exposure. Under such conditions, the Cd concentration in urine mainly reflects the amount of Cd stored in the body, particularly in the kidney. In Europe and the US, the Cd reference values are usually < 2 nmol/mmol creatinine. Because most of the Cd in urine is probably bound to metallothionein, the changes in the urinary metallothionein concentration parallel those of Cd. The determination of Cd concentration in hair is of limited value because in humans it is difficult to distinguish between externally deposited and endogenous Cd. Fecal Cd is a good indicator of the oral daily intake. The results of several cross-sectional epidemiologic studies of the relation between the prevalence of renal dysfunction and Cd concentration in urine led us to propose a biological limit value for Cd of 5 and 2 nmol/mmol creatine for adult male workers and the general population, respectively. (22 Refs.)

Tags: Human; Male

Descriptors: *Biological Markers--Analysis--AN; *Cadmium Poisoning; *Environmental Exposure; *Kidney Diseases--Chemically Induced--CI; Adult; Belgium; Cadmium--Analysis--AN; Cadmium--Blood--BL; Cadmium --Pharmacokinetics--PK; Cadmium--Urine--UR; Cadmium Poisoning--Prevention and Control--PC; Reference Values

CAS Registry No.: 0 (Biological Markers); 7440-43-9 (Cadmium)

16/9/10
DIALOG(R)File 155:MEDLINE(R)

07835052 94204607
Fatal cadmium-induced pneumonitis.
Seidal K; Jorgensen N; Elinder CG; Sjogren B; Vahter M
Department of Lung Medicine, Central Hospital, Karlstad, Sweden.
Scandinavian journal of work, environment & health (FINLAND) Dec 1993, 19 (6) p429-31, ISSN 0355-3140 Journal Code: UEB
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9407
Subfile: INDEX MEDICUS

A previously relatively healthy 78-year-old man was exposed to cadmium fumes during brazing with cadmium-containing silver solder. He developed severe chemical pneumonitis and died 25 d after exposure.

Tags: Case Report; Human; Male
Descriptors: *Bronchopneumonia--Chemically Induced--CI; *Cadmium Poisoning--Pathology--PA; *Pulmonary Fibrosis--Chemically Induced--CI; *Respiratory Insufficiency--Chemically Induced--CI; Aged; Alcoholic Beverages; Bronchopneumonia--Pathology--PA; Ethanol--Chemical Synthesis--CS ; Fatal Outcome; Leisure Activities; Pulmonary Fibrosis--Pathology--PA; Respiratory Insufficiency--Pathology--PA; Welding
CAS Registry No.: 64-17-5 (Ethanol)

16/9/11
DIALOG(R)File 155:MEDLINE(R)

07229124 93273400
Placental transfer of cadmium.
Lagerkvist BJ; Nordberg GF; Soderberg HA; Ekesrydh S; Englyst V; Gustavsson M; Gustavsson NO; Wiklund DE
Department of Environmental Medicine, Ume.ANG.a University, Sweden.
IARC scientific publications (FRANCE) 1992, (118) p287-91, ISSN 0300-5038 Journal Code: GKU
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9309
Subfile: INDEX MEDICUS
Cadmium levels in blood have been determined in mother-newborn pairs from the surroundings of a copper smelter and a control area in Northern Sweden. The smelter's cadmium emissions to the air have decreased substantially since the 1970s, and in 1989 the emission was one ton. Venous blood was sampled from the mothers during delivery and from the umbilical cords, and analysed for cadmium by flameless atomic absorption spectrophotometry. There were no significant differences in cadmium levels, as between exposed women and controls, and blood levels were low, even in an industrial area. The most important environmental exposure seemed to be smoking. There was, however, a significant increase in cadmium levels during pregnancy among non-smoking women in both groups, $p < 0.01$. The cadmium levels in the newborn babies were about 70% of those in the mothers. Cadmium levels in the babies of non-smoking mothers were significantly higher in the vicinity of the smelter than in the control area ($p < 0.05$).
Tags: Comparative Study; Female; Human; Support, Non-U.S. Gov't
Descriptors: *Cadmium--Pharmacokinetics--PK; *Environmental Exposure; *Infant, Newborn--Blood--BL; *Maternal-Fetal Exchange; *Placenta --Metabolism--ME; *Pregnancy Complications--Blood--BL; Cadmium--Blood--BL; Copper; Industry; Lead; Pregnancy; Pregnancy Complications--Chemically Induced--CI; Pregnancy Complications--Metabolism--ME
CAS Registry No.: 7439-92-1 (Lead); 7440-43-9 (Cadmium); 7440-50-8 (Copper)

16/9/12
DIALOG(R)File 155:MEDLINE(R)

07084505 92351094
Cadmium in blood and urine after cessation of exposure.
Gambini G; Leurini D
Divisione di Medicina del Lavoro, Ospedale Maggiore, Novara, Italy.
Science of the total environment (NETHERLANDS) Jun 9 1992, 120 (1-2) p111-5, ISSN 0048-9697 Journal Code: UJ0
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9211
Subfile: INDEX MEDICUS
A plant for the synthesis and packaging of cadmium oxide was operated for 32 months (1982-84) in a small chemical factory producing zinc and copper oxide. The cumulative exposure of 6 workers was from 12 to 190 days. Five years after cessation of exposure the blood cadmium levels in the exposed were, on average, 4-10 times higher than those of a reference group and the urinary cadmium levels were 1-6-times higher. In view of the long half-life of cadmium in the human body, the choice of normal reference values requires particular care since brief and sometimes forgotten exposures to cadmium may be a confounding factor to set reference values.
Tags: Human
Descriptors: *Cadmium; *Cadmium--Blood--BL; *Occupational Exposure; beta 2-Microglobulin--Metabolism--ME; beta 2-Microglobulin--Urine--UR; Cadmium --Urine--UR; Half-Life; Metabolic Clearance Rate; Reference Values; Time Factors
CAS Registry No.: 0 (beta 2-Microglobulin); 1306-19-0 (cadmium oxide) ; 7440-43-9 (Cadmium)

16/9/13
DIALOG(R)File 155:MEDLINE(R)

06686069 91209699
Transplacental transfer of cadmium and fetal effects.
Goyer RA
Department of Pathology, University of Western Ontario, London, Canada.
Fundamental and applied toxicology (UNITED STATES) Jan 1991, 16 (1) p22-3, ISSN 0272-0590 Journal Code: FAB
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9108
Subfile: INDEX MEDICUS
(6 Refs.)
Tags: Female; Human
Descriptors: *Cadmium--Pharmacokinetics--PK; *Cadmium Poisoning --Physiopathology--PP; *Fetus--Drug Effects--DE; Cadmium--Blood--BL; Cadmium--Toxicity--TO; Maternal-Fetal Exchange; Pregnancy
CAS Registry No.: 7440-43-9 (Cadmium)

16/9/14
DIALOG(R)File 155:MEDLINE(R)
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06488862 91020231
Transport of heavy metals by the kidney.
Foulkes EC
Department of Environmental Health, University of Cincinnati College of Medicine, OH 45267-0056.
Toxicology letters (NETHERLANDS) Sep 1990, 53 (1-2) p29-31, ISSN 0378-4274 Journal Code: VXN
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9101
Subfile: INDEX MEDICUS
(9 Refs.)
Tags: Animal; Human
Descriptors: *Kidney--Metabolism--ME; *Metals--Pharmacokinetics--PK; Biological Transport; Cadmium--Blood--BL; Cadmium--Pharmacokinetics--PK; Metals--Blood--BL
CAS Registry No.: 0 (Metals); 7440-43-9 (Cadmium)

16/9/15
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2000 Dialog Corporation. All rts. reserv.

05859781 90334965
Cadmium uptake kinetics in human erythrocytes.
Nguyen QH; Chien PK
Harney Science Center, Department of Biology, University of San Francisco, CA 94117-1080.
Biological trace element research (UNITED STATES) Nov 1989, 22 (2) p119-29, ISSN 0163-4984 Journal Code: AU1
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9011
Subfile: INDEX MEDICUS
Cross-membrane transport of cadmium in human erythrocytes was studied using 109Cd+(+) and liquid scintillation counting. Uptake rates were determined by depletion of radioactivity in the incubation medium and the amount of hemolyzate radioactivity taken up by the erythrocytes. Both saturable and nonsaturable components for cadmium transport were observed. The mean maximum uptake rate (Jmax) of the saturable component was 4.9 X 10(-6) mol/L/h. The transport constant (Kt) was estimated at 6.9 X 10(-5) mol/L. The diffusion constant (Kd) of the non-saturable component was 1.4 X 10(-2)/h. Both Jmax and Kt of cadmium generally decreased when Zn+(+) was present, with a biphasic response in the presence of Cu+(+). Kd of cadmium increased as Zn+(+) or Cu+(+) levels were increased. It is suggested that cadmium may penetrate human red cells via cation transport sites owing to its behavior as an analog of one or more nutrient species.
Tags: Human; In Vitro; Support, Non-U.S. Gov't
Descriptors: *Cadmium--Blood--BL; *Erythrocytes--Metabolism--ME; Biological Transport, Active; Cadmium--Pharmacokinetics--PK; Cadmium Radioisotopes--Diagnostic Use--DU; Copper--Blood--BL; Diffusion; Zinc --Metabolism--ME
CAS Registry No.: 0 (Cadmium Radioisotopes); 7440-43-9 (Cadmium); 7440-50-8 (Copper); 7440-66-6 (Zinc)

16/9/16
DIALOG(R)File 155:MEDLINE(R)

05594953 90019357
Cadmium concentration in human kidney biopsies.
Lindqvist B; Nystrom K; Stegmayr B; Wirell M; Eriksson A
Department of Internal Medicine, University Hospital, Ume.ANG.a, Sweden.
Scandinavian journal of urology and nephrology (SWEDEN) 1989, 23 (3) p213-7, ISSN 0036-5599 Journal Code: UDA
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9001
Subfile: INDEX MEDICUS
This study reports the cadmium concentration and histopathology of kidney tissue from 29 patients with clinical findings that motivated a diagnostic percutaneous kidney biopsy and/or a history of possible exposure to cadmium. Cadmium was found in all specimens examined including those from controls. In the patients, the mean cadmium concentration was 12.9 (0.6-45.0) micrograms cadmium per g wet kidney tissue. The highest concentrations (30-45 micrograms/g) were found in three patients with morphological and clinical findings of tubulo-interstitial damage. Patients with signs of tubulo-interstitial disease had higher mean cadmium concentrations than those with glomerular changes, and patients with normal blood pressure had higher concentrations than those with diastolic hypertension. In a control group of 22 autopsies, the mean cadmium concentration was 8.7 (2.9-22.4) micrograms/g. The mean difference between the right and the left kidney was 2.3 (0.9-9.6) micrograms/g. Laboratory findings in patients with cadmium nephropathy were nonspecific. Thus, in patients with interstitial nephritis and cadmium exposure, a biopsy for the analysis of kidney cadmium concentration may be motivated. The combination of morphological and clinical findings of interstitial nephritis and a high concentration of cadmium in biopsied kidney tissue indicates cadmium nephropathy.
Tags: Case Report; Female; Human; Male
Descriptors: *Cadmium--Analysis--AN; *Cadmium Poisoning--Pathology--PA; *Kidney--Analysis--AN; *Nephritis, Interstitial--Chemically Induced--CI; *Occupational Diseases--Chemically Induced--CI; Adult; Biopsy; Kidney --Pathology--PA; Middle Age; Nephritis, Interstitial--Pathology--PA; Occupational Diseases--Pathology--PA
CAS Registry No.: 7440-43-9 (Cadmium)

16/9/17
DIALOG(R)File 155:MEDLINE(R)

05351106 88305019
Warning against the indiscriminate use of a biological half-time model in deriving the critical concentration of metals.
Sugita M; Tsuchiya K
Department of Public Health, School of Medicine, Tokai University, Isehara, Japan.
Sangyo Ika Daigaku zasshi (JAPAN) Jun 1 1988, 10 (2) p179-88, ISSN 0387-821X Journal Code: SID
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8811
Subfile: INDEX MEDICUS
For heavy metals, many studies obtained short biological half-times (BHTs) by administrations of heavy metals. Tsuchiya and Sugita, however, first reported the possibility of a long BHT for cadmium (Cd) calculated from Cd accumulations in postmortem human organs and tissues by age using a non-linear regression method employing a differential equation. According to their reports, the Cd BHTs (point estimators) were 12.1-22.7 years by sex, renal cortex and medulla. The minimums and the maximums of the Cd BHTs on the 95% confidence regions of estimators were 6.9-70.2 years by sex and kidney part. It is presumed that the range of the 95% confidence region for the individual BHTs of renal Cd exists in a range from a few years to at least 100 years because of large individual variations in exposure, absorption rate and excretion rate. Point estimators of BHTs, however, include the assumption that all subjects have been exposed to the same level of Cd at the same year of age over a period of decades and have equal absorption and excretion rates of Cd. Therefore, it is not adequate to calculate a safety level for Cd in the industrial environment or foodstuffs using a value of Cd BHT (point estimator) based on Cd accumulation applying a mathematical model. BHTs of metals require careful evaluation and must not be used indiscriminately to derive a critical concentration, for example, using a mathematical model.
Tags: Female; Human; Male
Descriptors: *Cadmium--Pharmacokinetics--PK; *Kidney--Metabolism--ME; Adolescence; Adult; Age Factors; Aged; Aged, 80 and over; Child; Child, Preschool; Half-Life; Infant; Middle Age; Models, Biological; Regression Analysis
CAS Registry No.: 7440-43-9 (Cadmium)

16/9/18
DIALOG(R)File 155:MEDLINE(R)
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05227548 86275198
Cadmium in human population.
Bernard A; Lauwerys R
Experientia. Supplementum (SWITZERLAND) 1986, 50 p114-23, ISSN 0071-335X Journal Code: EQ1
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW
JOURNAL ANNOUNCEMENT: 8611
Subfile: INDEX MEDICUS
(104 Refs.)
Tags: Animal; Human
Descriptors: *Cadmium--Analysis--AN; *Cadmium Poisoning; *Environmental Exposure; Cadmium--Metabolism--ME; Cell Transformation, Neoplastic--Drug Effects--DE; Electrophoresis, Agar Gel; Half-Life; Hypertension--Etiology --ET; Intestinal Absorption; Kidney--Analysis--AN; Kidney--Drug Effects --DE; Liver--Analysis--AN; Liver--Drug Effects--DE; Lung--Analysis--AN; Respiration; Smoking; Tissue Distribution; Tobacco
CAS Registry No.: 7440-43-9 (Cadmium)

16/9/19
DIALOG(R)File 155:MEDLINE(R)
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05218602 86166539
Cadmium metabolism in man.
Kelman GR
Human toxicology (ENGLAND) Mar 1986, 5 (2) p91-3, ISSN 0144-5952 Journal Code: GFR
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8607
Subfile: INDEX MEDICUS
The time course of the fall in blood cadmium concentrations after cessation of exposure has been measured in nine workmen exposed to cadmium. When the initial blood cadmium concentration was below 180 nmol/l (six subjects) it declined smoothly and roughly exponentially, with a mean half-life of 20.4 months and a final asymptote of 70 nmol/l; in the remaining three subjects (initial blood cadmium concentration above 180 nmol/l) the decay was less regular and more prolonged (mean half-life 31.4 months, final asymptote 92 nmol/l). The significance of these results in relation to occupational cadmium exposure is discussed.
Tags: Human; Male
Descriptors: *Cadmium--Blood--BL; beta 2-Microglobulin--Urine--UR; Adult; Aged; Air Pollutants, Occupational--Analysis--AN; Cadmium--Urine--UR; Half-Life; Middle Age; Prospective Studies; Smoking; Time Factors
CAS Registry No.: 0 (beta 2-Microglobulin); 0 (Air Pollutants, Occupational); 7440-43-9 (Cadmium)

16/9/20
DIALOG(R)File 155:MEDLINE(R)

05069884 87299188
Cadmium-induced osteopathy: clinical and autopsy findings of four patients.
Takebayashi S; Harada T; Kamura S; Satoh T; Segawa M; Yajima K
Applied pathology (SWITZERLAND) 1987, 5 (3) p190-7, ISSN 0252-1172
Journal Code: APP
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8712
Subfile: INDEX MEDICUS
Clinical and autopsy findings of 4 patients with chronic cadmium toxication by peroral uptake of cadmium are reported. Cadmium toxication was liable to occur in multiparous postmenopausal women, and it began with proteinuria, glycosuria, lumbago and bone pain. Then, renal function gradually decreased being accompanied with renal tubulopathy. Autopsy disclosed renal tubulopathy, which consisted of the flattening of the epithelium of proximal convoluted tubules at the peripheral portion and the mild thickening of the tubular basement membrane. There was no primary change in the glomerulus and renal interstitium. Osteomalacia was observed in the vertebrae and several other bones. The degree of osteomalacia was in good agreement with chronic renal tubular dysfunction. A decrease of the estrogen content, in addition to renal tubulopathy due to biological saturation of cadmium, seems to play an important role in the pathogenesis of cadmium-induced osteomalacia.
Tags: Case Report; Female; Human; Male
Descriptors: *Bone and Bones--Pathology--PA; *Cadmium Poisoning --Pathology--PA; *Osteomalacia--Chemically Induced--CI; Acidosis, Renal Tubular--Chemically Induced--CI; Acidosis, Renal Tubular--Pathology--PA; Aged; Aged, 80 and over; Kidney Tubules--Pathology--PA; Osteomalacia --Pathology--PA

16/9/21
DIALOG(R)File 155:MEDLINE(R)

04213866 85015707
Molecular basis of cadmium toxicity.
Nath R; Prasad R; Palinal VK; Chopra RK
Progress in food & nutrition science (ENGLAND) 1984, 8 (1-2) p109-63,
ISSN 0306-0632 Journal Code: Q08
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW
JOURNAL ANNOUNCEMENT: 8501
Subfile: INDEX MEDICUS

Cadmium has been shown to manifest its toxicity in human and animals by mainly accumulating in almost all of the organs and kidney is the main target organ where it is concentrated mainly in cortex. Environmental exposure of cadmium occurs via food, occupational industries, terrestrial and aquatic ecosystem. At molecular level, cadmium interferes with the utilization of essential metals e.g. Ca, Zn, Se, Cr and Fe and deficiencies of these essential metals including protein and vitamins, exaggerate cadmium toxicity, due to its increased absorption through the gut and greater retention in different organs as metallothionein (Cd-Mt). Cadmium transport, across the intestinal and renal brush border membrane vesicles, is carrier mediated and it competes with zinc and calcium. It has been postulated that cadmium shares the same transport system. Cadmium inhibits protein synthesis, carbohydrate metabolism and drug metabolizing enzymes in liver of animals. Chronic environmental exposure of cadmium produces hypertension in experimental animals. Functional changes accompanying cadmium nephropathy include low molecular weight proteinuria which is of tubular origin associated with excess excretion of proteins such as beta 2 microglobulin, metallothionein and high molecular weight proteinuria of glomerular origin (excretion of proteins such as albumin IgG, transferrin etc.). Recent data has shown that metallothionein is more nephrotoxic to animals. Cadmium is also toxic to central nervous system. It causes an alterations of cellular functions in lungs. Cadmium affects both humoral and cell mediated immune response in animals. Cadmium induces metallothionein in liver and kidney but under certain nutritional deficiencies like protein-calorie malnutrition and calcium deficiency, enhanced induction and greater accumulation of cadmium metallothionein has been observed. (380 Refs.)

Tags: Animal; Female; Human; Male; Support, Non-U.S. Gov't
Descriptors: *Cadmium--Adverse Effects--AE; Aging; Bone Diseases --Chemically Induced--CI; Cadmium--Blood--BL; Cadmium--Metabolism--ME; Calcium--Metabolism--ME; Central Nervous System Diseases --Chemically Induced--CI; Chromium--Metabolism--ME; Copper--Metabolism--ME; Dietary Proteins--Pharmacology--PD; Drug Interactions; Environmental Exposure; Half-Life; Hypertension--Chemically Induced--CI; Immunity--Drug Effects--DE ; Intestinal Absorption; Intestinal Diseases--Chemically Induced--CI; Iron --Metabolism--ME; Kidney Diseases--Chemically Induced--CI; Liver --Drug Effects--DE; Lung--Drug Effects--DE; Metallothionein--Physiology--PH; Ovary --Drug Effects--DE; Selenium--Metabolism--ME; Sex Factors; Testis--Drug Effects--DE; Tissue Distribution; Vitamins--Metabolism--ME; Zinc --Metabolism--ME
CAS Registry No.: 0 (Dietary Proteins); 0 (Vitamins); 7439-89-6 (Iron); 7440-43-9 (Cadmium); 7440-47-3 (Chromium); 7440-50-8 (Copper) ; 7440-66-6 (Zinc); 7440-70-2 (Calcium); 7782-49-2 (Selenium); 9038-94-2 (Metallothionein)

16/9/22
DIALOG(R)File 155:MEDLINE(R)

03963501 84123713
An acute fatal occupational cadmium poisoning by inhalation.
Yamamoto K; Ueda M; Kikuchi H; Hattori H; Hiraoka Y
Zeitschrift fur Rechtsmedizin. Journal of legal medicine (GERMANY, WEST) 1983, 91 (2) p139-43, ISSN 0044-3433 Journal Code: Y0N
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8405
Subfile: INDEX MEDICUS

A 43-year-old male smelter was admitted to a hospital on account of severe dyspnea about 2 days after exposure to brownish-yellow smoke produced by melting of "copper" scrap. On admission pronounced hypoxemia was revealed, and an oxygen-enriched gas was administered after intubation. Although inspired oxygen concentration was gradually increased, hypoxemia progressed and he died on day 11 in hospital. The principal autopsy finding was chiefly confined to the lungs. Both lungs were heavy (the left weighing 1,470 g; the right 1,710 g) and firm to the touch. Histologically, no normal alveoli were found throughout the entire lung. Some alveolar spaces were occupied by pneumocytes, others by organized exudate with fibrosis. Interstitial fibrosis was present. Patchy areas of inflammatory cell infiltrations as well as intra-alveolar hemorrhages were observed. On the basis of the above findings a diagnosis of diffuse alveolar damage was made. Based on the available evidence (presence of cadmium in the "copper" scrap, feature of the smoke, clinical signs with latent time, and high cadmium concentration of the lung), the diffuse alveolar damage was considered to have been caused by inhaled cadmium. The pulmonary change of the present case was more advanced in pathologic stage in comparison with those reported in the literature.

Tags: Case Report; Human; Male
Descriptors: *Cadmium Poisoning--Pathology--PA; *Forensic Medicine; *Occupational Diseases--Chemically Induced--CI; Adult; Environmental Exposure; Lung--Drug Effects--DE; Lung--Pathology--PA; Metallurgy; Occupational Diseases--Pathology--PA; Pulmonary Fibrosis --Chemically Induced--CI; Pulmonary Fibrosis--Pathology--PA

16/9/23
DIALOG(R)File 155:MEDLINE(R)

03935920 84044638
Biological half-time of cadmium in the blood of workers after cessation of exposure.
Jarup L; Rogenfelt A; Elinder CG; Nogawa K; Kjellstrom T
Scandinavian journal of work, environment & health (FINLAND) Aug 1983, 9 (4) p327-31, ISSN 0355-3140 Journal Code: UEB
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8402
Subfile: INDEX MEDICUS
The biological half-time of cadmium in the blood of previously exposed workers was estimated after the cessation of exposure. Five men were followed for a period of 10 to 13 years. One-compartment and two-compartment exponential elimination models were used to describe the decrease in blood cadmium levels over time. The best fit to the observed data was obtained with a two-compartment model. The half-times estimated from this model ranged from 75 to 128 d for the fast component and from 7.4 to 16.0 years for the slow component. The results confirm that there is a very long whole-body biological half-time for cadmium, and the estimated half-times are similar to those obtained with different methods.
Tags: Human; Male
Descriptors: *Cadmium--Blood--BL; *Environmental Exposure; *Occupational Medicine; Adult; Aged; Follow-Up Studies; Half-Life; Middle Age
CAS Registry No.: 7440-43-9 (Cadmium)

16/9/24
DIALOG(R)File 155:MEDLINE(R)

03815508 81044064
Multispecies retention parameters for cadmium.
Thomas RG; Wilson JS; London JE
Environmental research (UNITED STATES) Oct 1980, 23 (1) p191-207, ISSN 0013-9351 Journal Code: EI2
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8103
Subfile: INDEX MEDICUS
Tags: Animal; Female; Human; Male; Support, U.S. Gov't, Non-P.H.S.
Descriptors: *Cadmium--Metabolism--ME; Body Burden; Cadmium --Administration and Dosage--AD; Cadmium--Diagnostic Use--DU; Dogs; Half-Life; Haplorhini; Mice; Radioisotopes--Diagnostic Use--DU; Rats; Species Specificity; Tissue Distribution
CAS Registry No.: 0 (Radioisotopes); 7440-43-9 (Cadmium)

16/9/25
DIALOG(R)File 155:MEDLINE(R)

03765474 81230903
Cadmium nephropathy.
Chan WY; Rennert OM
Annals of clinical and laboratory science (UNITED STATES) May-Jun 1981, 11 (3) p229-38, ISSN 0091-7370 Journal Code: 532
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW
JOURNAL ANNOUNCEMENT: 8110
Subfile: INDEX MEDICUS

Cadmium, an important environmental toxic agent has the kidney as its most important target organ. It is concentrated mainly in the renal cortex. Excessive renal accumulation of cadmium causes well defined morphological and ultrastructural pathological changes in the proximal tubules. Functional changes accompanying cadmium nephropathy include proteinuria, enzymuria, aminoaciduria, glycosuria, polyuria, hepercalciuria, increased urinary uric acid, and cadmium. The observed proteinuria has two components: low molecular weight proteinuria of tubular origin (excess excretion of proteins such as B2-microglobulin) and high molecular weight proteinuria of glomerular origin, (excretion of proteins such as albumin, IgG, transferrin, etc.) The proposed mechanisms of cadmium nephropathy are reviewed. The involvement of metallothionein in cadmium nephropathy and the nephrotoxic effects of cadmium-thionein are discussed. (89 Refs.)

Tags: Animal; Human
Descriptors: *Cadmium Poisoning--Pathology--PA; *Kidney Diseases --Pathology--PA; Cadmium--Metabolism--ME; Cadmium Poisoning--Metabolism--ME ; Cadmium Poisoning--Physiopathology--PP; Carrier Proteins--Metabolism--ME ; Kidney--Metabolism--ME; Kidney--Pathology--PA; Kidney--Physiopathology --PP; Kidney Tubules, Proximal--Pathology--PA; Metallothionein--Metabolism --ME; Metallothionein--Toxicity--TO
CAS Registry No.: 0 (cadmium-binding protein); 0 (Carrier Proteins); 7440-43-9 (Cadmium); 9038-94-2 (Metallothionein)

16/9/26
DIALOG(R)File 155:MEDLINE(R)

03536792 82181213
Additive statistical effects of cadmium and lead on heart-related disease in a North Carolina autopsy series.
Voors AW; Shuman MS; Johnson WD
Archives of environmental health (UNITED STATES) Mar-Apr 1982, 37 (2) p98-102, ISSN 0003-9896 Journal Code: 6YO
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8208
Subfile: AIM; INDEX MEDICUS
The association of heart-related mortality with tissue cadmium and lead in a study of autopsies performed on persons who resided in a soft-water, leached-soil area of North Carolina was examined. Liver cadmium concentrations and aortic lead level were indices of these elements. Both cadmium and lead levels had statistically significant correlations with cause of death (heart-related disease vs. non-heart-related disease, excluding cancer). Although cause of death was significantly associated with age, the association with cadmium and lead persisted after statistical adjustment for the effect of age. The combined effects of cadmium and lead provided sufficient information in an additive model to predict cause of death correctly for 80% of the cases, with age contributing insignificantly. These findings indicate the intimate relation of these two trace metals with increased risk of heart-related mortality, even in light of known conventional causes of such deaths.
Tags: Female; Human; Male; Support, U.S. Gov't, Non-P.H.S.
Descriptors: *Cadmium Poisoning--Pathology--PA; *Cardiovascular Diseases --Etiology--ET; *Lead Poisoning--Pathology--PA; Adult; Aged; Aorta, Abdominal--Analysis--AN; Cadmium--Analysis--AN; Cadmium Poisoning --Complications--CO; Cardiovascular Diseases--Mortality--MO; Environmental Exposure; Lead--Analysis--AN; Lead Poisoning--Complications--CO; Liver --Analysis--AN; Middle Age; North Carolina; Regression Analysis
CAS Registry No.: 7439-92-1 (Lead); 7440-43-9 (Cadmium)

16/9/27
DIALOG(R)File 155:MEDLINE(R)

03480737 82072360
Metabolism of orally ingested cadmium in humans.
Shaikh ZA; Smith JC
Developments in toxicology and environmental science (NETHERLANDS) 1980 , 8 p569-74, ISSN 0165-2214 Journal Code: ECX
Contract/Grant No.: ES 01247, ES, NIEHS; ES 01248, ES, NIEHS
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8204
Subfile: INDEX MEDICUS
Tags: Female; Human; Male; Support, U.S. Gov't, P.H.S.
Descriptors: *Cadmium--Metabolism--ME; Administration, Oral; Adolescence; Adult; Feces--Analysis--AN; Half-Life; Intestinal Absorption; Radioisotopes
CAS Registry No.: 0 (Radioisotopes); 7440-43-9 (Cadmium)

16/9/28
DIALOG(R)File 155:MEDLINE(R)

02837356 89377725

Cadmium concentrations in human liver, blood, and bile: comparison with a metabolic model.

Elinder CG; Kjellstom T; Lind B; Molander ML; Silander T

Department of Environmental Hygiene, Karolinska Institute, Stockholm, Sweden.

Environmental research (UNITED STATES) Oct 1978, 17 (2) p236-41, ISSN 0013-9351 Journal Code: EI2

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 8912

Subfile: INDEX MEDICUS

Cadmium concentrations in liver biopsies, blood, and bile were measured by atomic absorption spectrophotometry in 23 patients in connection with routine gallstone operations. On a group basis cadmium in blood was a good indicator of cadmium in liver, and the estimated linear relationship agreed well with calculations from a formerly proposed metabolic model. Cadmium in bile was also analyzed, and an average of about 2.5 ng of Cd/g wet weight was found. This is about 10 times more than would have been expected form the metabolic model and suggests that bile might be an important excretion route for cadmium. Definite conclusions cannot be drawn, however, since the results could not be cross-checked with neutron activation analysis, due to insufficient sensitivity of the latter method.

Tags: Comparative Study; Female; Human; Male

Descriptors: *Bile--Metabolism--ME; *Cadmium--Analysis--AN; *Liver --Metabolism--ME; *Models, Biological; Adult; Aged; Bile--Drug Effects--DE; Cadmium--Blood--BL; Cadmium--Pharmacokinetics--PK; Liver--Drug Effects--DE ; Metabolic Clearance Rate; Middle Age; Spectrophotometry, Atomic Absorption

CAS Registry No.: 7440-43-9 (Cadmium)

16/9/29
DIALOG(R)File 155:MEDLINE(R)

02677994 80024158
Metabolic model for cadmium in man.
Nordberg GF; Kjellstrom T
Environmental health perspectives (UNITED STATES) Feb 1979, 28 p211-7, ISSN 0091-6765 Journal Code: EI0
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8002
Subfile: INDEX MEDICUS
A metabolic model for cadmium has been formulated in terms of a flow scheme for cadmium among eight body compartments. The mathematical description of the flow of cadmium between compartments consits of a number of differential equations, and the accumulation of cadmium may thus be calculated. Coeffcients for the flow of cadmium were estimated from empirical data both from animals and man. The modelling serves two main purposes: it provides a means of using present knowledge about metabolism in order to calculate expected accumulation in critical organs and other tissues and body fluids at certain intake levels and it makes it possible to define deficiencies in our present knowledge about metabolism. Data recently collected, partly as a result of considerations related to the model, have confirmed the assumptions of a very long biological half-time in other tissues and the small excretion of cadmium via bile.
Tags: Human
Descriptors: *Cadmium--Metabolism--ME; *Models, Biological; Adult; Age Factors; Air Pollution; Cadmium--Blood--BL; Cadmium--Urine--UR; Environmental Exposure; Evaluation Studies; Food Contamination; Half-Life; Industry; Kidney Cortex--Metabolism--ME; Kinetics; Middle Age; Smoking; Tissue Distribution

16/9/30
DIALOG(R)File 155:MEDLINE(R)
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02504312 78149112
Increased dietary cadmium absorption in mice and human subjects with iron deficiency.
Flanagan PR; McLellan JS; Haist J; Cherian G; Chamberlain MJ; Valberg LS
Gastroenterology (UNITED STATES) May 1978, 74 (5 Pt 1) p841-6, ISSN 0016-5085 Journal Code: FH3
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 7808
Subfile: AIM; INDEX MEDICUS
In mice fed a low iron diet, the addition of low levels of cadmium chloride (10 micrometer) to the drinking water impaired growth and accentuated the development of anemia. Cadmium had no effect on mice given a similar diet supplemented with iron. Iron deficiency increased the concentration of cadmium in the duodenal mucosa, the transfer of cadmium to the body from the intestinal tract, and the deposition of absorbed cadmium in the kidneys. In human subjects, the average absorption of 25 microgram of cadmium, labeled with 115mCd, from a test meal was 8.9 +/- 2.0% (mean +/- SE) in 10 people with low body iron stores (serum ferritin less than 20 ng per ml) and 2.3 +/- 0.3% in 12 subjects with normal iron stores (serum ferritin greater than 23 ng per ml). The biological half-time of the radiocadmium in 3 of the subjects ranged from 90 to 202 days. Thus, the intestinal adaptive response to iron deficiency in both experimental animals and human subjects leads to the increased absorption of cadmium, a potentially toxic element.
Tags: Animal; Female; Human
Descriptors: *Cadmium--Metabolism--ME; *Iron--Deficiency--DF; Anemia, Hypochromic--Etiology--ET; Body Burden; Body Weight; Diet; Drinking; Duodenum--Metabolism--ME; Ferritin--Blood--BL; Half-Life; Hematocrit; Intestinal Absorption; Intestinal Mucosa--Metabolism--ME; Kidney --Metabolism--ME; Liver--Metabolism--ME; Mice; Mice, Inbred C57BL; Solubility

16/9/31
DIALOG(R)File 155:MEDLINE(R)
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02214781 77075014
Mathematical derivation of the biological half-time of cadmium in human organs based on the accumulation of the metal in the organs.
Tsuchiya K; Sugita M; Seki Y
Keio journal of medicine (JAPAN) Apr 1976, 25 (2) p73-82, ISSN 0022-9717 Journal Code: KUJ
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 7704
Subfile: INDEX MEDICUS
Tags: Human; Male
Descriptors: *Cadmium--Metabolism--ME; *Models, Biological; Adolescence; Adult; Aged; Child; Child, Preschool; Half-Life; Infant; Infant, Newborn; Kidney--Metabolism--ME; Middle Age

16/9/32
DIALOG(R)File 155:MEDLINE(R)

02210843 77064598
Cadmium in kidney cortex, liver, and pancreas from Swedish autopsies. Estimation of biological half time in kidney cortex, considering calorie intake and smoking habits.
Elinder CG; Lind B; Kjellstrom T; Linnman L; Friberg L
Archives of environmental health (UNITED STATES) Nov-Dec 1976, 31 (6) p292-302, ISSN 0003-9896 Journal Code: 6YO
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 7703
Subfile: AIM; INDEX MEDICUS
Cadmium and zinc have been analyzed in tissues from 292 persons autopsied in Stockholm. In kidney cortex, liver, and pancreas the individual cadmium levels are distributed in a lognormal way. In kidney cortex there is a continuous accumulation of cadmium with age up to 50 years, followed by a decrease. Smokers show a higher cadmium accumulation. For nonsmokers, the biological half time of cadmium in kidney cortex is estimated at 30 years, with an average concentration at age 50 of 11 mug/g wet weight. When smokers are included, the average cadmium concentration at age 50 is 22 mug/g wet weight. Based on the more pronounced cadmium accumulation among smokers than nonsmokers, the respiratory absorption rate of cadmium from tobacco smoke is estimated to be approximately 50%.
Tags: Female; Human; Male
Descriptors: *Cadmium--Metabolism--ME; *Kidney Cortex--Metabolism--ME; *Liver--Metabolism--ME; *Pancreas--Metabolism--ME; *Smoking; Adolescence; Adult; Age Factors; Aged; Child; Child, Preschool; Half-Life; Infant; Infant, Newborn; Middle Age; Sweden; Zinc--Metabolism--ME

16/9/33
DIALOG(R)File 155:MEDLINE(R)

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Table 3. Citations of Possible Interest

Lauwerys, R. Buchet, J. P., Roels, H., Bernard, A., Chettle, D.R., Harvey, T. C., & Al Haddad, I.K. (1980) Biological signficance of cadmium concentration in blood and urine and their application in monitoring workers exposed to cadmium. In :*Edited Proceedings of the Second International Cadmium Conference*, Cannes. 6-8 February 1979, London, Metal Bulletin Ltd. Pp. 164-167

Sheng, S.L., Balakrishnan, N., Chettle, D.R., Classical and Bayesian Approaches to Compartment Models Based on in vivo Cadmium Data, Mathematical and Computer Modelling 32 (2000) 273-288.